U.S. Department of Labor

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Issue Date: 08 September 2004 In The Matter Of

CASE NOS.: 2004-BLA-05423
2004-BLA-00154

MARIAN L. ISRAEL (O/B/O AND WIDOW OF FREDERICK JOHN ISRAEL)
Claimant

v.

GILBERTON ENERGY CORP.
Employer
And

SECURITY INS. CO. OF HARTFORD
Carrier

And

DIRECTOR, OFFICE OF WORKERS' COMPENSATION PROGRAMS
Party-in-Interest

APPEARANCES:

Helen M. Koschoff, Esq.
For the Claimant

James M. Poerio, Esq.
For the Employer

Before: PAUL H. TEITLER,
Administrative Law Judge

DECISION AND ORDER - DENYING BENEFITS

This proceeding arises from a claim for benefits under the Black Lung Benefits Act of 1977, 30 U.S.C. § 901, et seq. by the deceased miner which has been consolidated with a claim for survivor's benefits filed under the Act by the deceased miner's widow. In accordance with the Act and the regulations issued thereunder, the two cases were referred by the Director, Office

of Workers' Compensation Programs to the Office of Administrative Law Judges for a formal hearing.

Benefits under the Act are awarded to miners who are totally disabled within the meaning of the Act due to pneumoconiosis, or to the survivors of miners whose deaths were caused by pneumoconiosis. Pneumoconiosis is a dust disease of the lungs arising from coal mine employment and is commonly known as "black lung".

A formal hearing was held before the undersigned on April 14, 2004, in Reading, Pennsylvania at which all parties were afforded full opportunity, in accordance with the Rules of Practice and Procedure (29 C.F.R. Part 18), to present evidence and argument as provided in the Act and the regulations issued thereunder, set forth in Title 20, Code of Federal Regulations, Parts 410, 718, 725, and 727. Claimant and Employer were represented by counsel. No appearance was entered on behalf of the Director, Office of Workers' Compensation Programs. Since Marion Israel, the Claimant-widow, had testified at the hearing before me in the miner's claim on July 12, 2001 (DX 38), she did not testify at the most recent hearing.

At the hearing, Director's Exhibits 1 through 65 which included Director's Exhibit 1 through 39 for the miner's claim and Director's Exhibits 40 through 65 in the widow's claim were identified. In addition, Claimant's Exhibits 1 through 5 and Employer's Exhibits 1 through 4 were identified. I omitted, however, entering these exhibits into the record and they are hereby entered into the record as identified at the hearing. In addition, subsequent to the hearing, pursuant to discussion at the hearing (TR 14, 18) Employer submitted the transcript of a deposition taken on April 7, 2004 of Dr. S. Levinson which is hereby entered into evidence as Employer' Exhibit 5. Subsequent to the hearing, both parties requested enlargement of time or continuance to submit additional evidence as discussed at the hearing. Each party objected to the request of the other party. By Order dated June 8, 2004, I stated I would not allow for the June 14, 2004 closing date to be extended and denied Respondent's request for a continuance. By Order dated June 16, 2004, I denied Claimant's motion for enlargement of time. In addition, neither party has submitted a post-hearing brief. Accordingly, the record is now closed[1].

Procedural History

In the deceased miner's second claim for benefits, in a Decision and Order on Remand Denying Benefits issued on January 10, 1996, Administrative Law Judge Brown found the presence of pneumoconiosis which arose out of coal mine employment had been established. Judge Brown found, however, that total disability due to pneumoconiosis has not been established (DX 26-77). Claimant subsequently appealed the denial to the Benefits Review Board (Board). After an Order to Show Cause from the Board why the appeal should not be dismissed for Claimant's failure to submit a petition for review, Claimant then requested remand to the District Director to pursue modification proceedings. On October 24, 1996, the Board granted Claimant's request and remanded the matter to the District Director for modification

[1] The following abbreviations will be used when citing to the record: DX - Director's Exhibits; CX – Claimant's Exhibits; EX - Employer's Exhibits; and TR - Transcript of Hearing of April 14, 2004.

proceedings (DX 26-85). On December 4, 1997, however, Administrative Law Judge Brown dismissed Claimant's request for modification and noted Claimant's failure to respond to Employer's motion to dismiss the claim for Claimant's failure to comply with reasonable discovery requests (DX 26).

On December 3, 1999, Claimant-miner filed his third claim for benefits (DX 1). This claim was denied by the Office of Workers' Compensation Programs on August 9, 2000 (DX 19). Claimant requested a hearing on August 22, 2000 (DX 20). By letter dated February 15, 2001, the widow stated it was her wish to pursue the miner's claim. The miner had died on September 2, 2000 (DX 25). The miner's case was then referred to this Office, however, on April 26, 2002, following a hearing on July 12, 2001, the undersigned remanded the miner's claim to be consolidated with the widow's claim (DX 39).

The widow, Marion Israel, had applied for survivor's benefits on January 3, 2002 (DX 41). Her claim was denied on September 3, 2003 by the District Director who found the presence of pneumoconiosis which arose out of coal mine employment had been established. However, since the evidence did not establish that the miner's death was due to pneumoconiosis, the widow's claim for survivor's benefits was denied (DX 60). On September 5, 2003, the widow requested a hearing (DX 61). The claims were consolidated and were referred to this Office on December 12, 2003 (DX 63). As noted above, a hearing was held on April 14, 2004.

In a Decision and Order issued on May 23, 1994 on the miner's second claim for benefits, Judge Brown found forty-six years of coal mine employment had been established (DX 35). This finding was affirmed by the Board in a Decision and Order issued on January 31, 1996 (DX 26-76). In addition, Judge Brown found the presence of pneumoconiosis which arose out of coal mine employment had been established (DX 35, DX 26-77). That finding is well supported by the recently submitted anatomical evidence. Judge Brown found, however, that the evidence did not establish the miner was totally disabled by pneumoconiosis. In addition, in the widow's claim the Employer contends the evidence does not establish the miner's death was due to pneumoconiosis and, therefore, the widow's claim should be denied.

Based on an analysis of the entire record, including the transcripts, exhibits, and representations of the parties, with consideration being given to the applicable statutory provisions, regulations and case law, I make the following:

FINDINGS OF FACT AND CONCLUSIONS OF LAW

Medical Evidence

The prior descriptions of medical evidence set forth in Judge Brown's determinations of May 23, 1994 and June 10, 1996 are hereby incorporated by reference (DX 22, 58). In addition, the following additional evidence has been submitted:

Chest X-Ray Reports

EX. NO.	DOCTOR[2]	DATE OF X-RAY	READING
DX 26-91	Levinson	03-06-97	0/1 q, q, co, ef
DX 26-93	Wolfe, B/BCR	03-06-97	No pneumoconiosis, co, ef
DX 8	Barrett, B/BCR	01-21-00	No pneumoconiosis, co, ef
DX 16, 17, 30	Wolfe, B/BCR	01-21-00	No pneumoconiosis, emphysema
DX 29	Levinson	05-25-00	0/1 q, q, emphysema

Pulmonary Function Studies

EX. NO.	PHYSICIAN	DATE	AGE	FEV_1	FEV_1 FVC	FVC	MVV
DX 47	Bane	09-03-91	64	3.03	3.74	81%	30
DX 26-91	Levinson	03-06-97	70	1.65	1.91	86%	45
				1.42	2.07	69%	47
DX 44	Kraynak	11-24-97	70	1.52	1.69	90%	52
DX 2	Mariglio	01-21-00	72	1.64	2.24	73%	36
				1.45	2.16	67%	41

In addition, Dr. Levinson testified about a pulmonary function study he performed on July 10, 2000, however, neither the report nor the tracings are included in the record. Employer has submitted extensive documentation establishing that copies were sent to the Claimant's counsel (EX 4), however, the report and tracings are not included in the record. Dr. Levinson testified the Claimant demonstrated a 1.97 on the FEV-1 maneuver and a 2.61 on the FVC maneuver with only fair effort on July 10, 2000 (EX 5).

[2] The symbol "B" denotes a physicians who was an approved "B-reader" at the time of the x-ray reading. A B-reader is a radiologist who has demonstrated his expertise in assessing and classifying x-ray evidence of pneumoconiosis. These physicians have been approved as proficient readers by the National Institute of occupational Safety & Health, U.S. Public Health Service pursuant to 42 C.F.R. §37.51 (1982). The symbol "BCR" denotes a physician who has been certified in radiology or diagnostic roentgenology by the American Board of Radiology, Inc., or the American Osteopathic Association. 20 C.F.R. §727.206(b)(2)(III).

Blood Gas Studies

EX. NO.	DATE	DOCTOR	pCO_2	pO_2
DX 26-91	03-15-1997	Levinson	31	90
DX 4	01-21-2000	Mariglio	31 29	69 (at rest) 56 (after exercise)
DX 29	05-26-2000	Levinson	34	68

Physician Examination Reports

On February 25, 2000, Dr. J. Mariglio examined Claimant-miner and reported normal findings on inspection, palpation and percussion with fine rales at the bases and rhonchi in the right base on auscultation. Dr. Mariglio performed a chest x-ray, pulmonary function study and blood gas study. He diagnosed coal worker's pneumoconiosis based on the results of physical examination, chest x-ray, exercise blood gas study results and pulmonary function study results. Dr. Mariglio also noted the miner had arteriosclerotic heart disease, status-post coronary artery bypass graft, and peripheral vascular disease. He stated the coal worker's pneumoconiosis contributed significantly to the laboratory changes. Dr. Mariglio did not, however, include any discussion of the basis of these conclusions (DX 3).

On June 12, 2000, Dr. G. Fino, reviewed the examination findings of February 25, 2000 and the pulmonary function study of January 21, 2000. He stated, relying on his previous medical evidence reviews and the review of this evidence, there is no evidence the miner has a coal mine dust related pulmonary condition (DX 17, 31).

Dr. S. Levinson, a pulmonary specialist, examined the miner on March 6, 1997 and performed various pulmonary studies. He reported there was no evidence of industrial pulmonary disease. Dr. Levinson also found no evidence of a disability from a pulmonary standpoint, however, he noted the miner's significant arteriosclerotic cardiovascular disease with a history of prior myocardial infarction (DX 26-91).

On May 25, 2000, Dr. Levinson, examined the miner a second time. He noted the miner's extensive medical history, coal mine employment history of 45 years and smoking history from 1946 to 1991. Dr. Levinson reported on examination the lungs were clear to auscultation and percussion. He reported the x-ray film was negative for pneumoconiosis but did show evidence of emphysema and previous heart surgery. On pulmonary function studies, he reported the miner's results were better than March of 1997 despite only fair effort. On blood gas studies, Dr. Levinson reported a slight increase in the carboxyhemoglobin values which indicated the miner was still smoking. Dr. Levinson also reported a mild hypoxemia on blood gas study. He concluded the miner was not suffering from a pulmonary disease. He stated there is evidence of chronic obstructive pulmonary disease and pulmonary emphysema due to cigarette

smoking as well as a right lower lobe density and right pleural effusion which were suggestive of malignancy. Dr. Levinson noted the miner's advance arteriosclerotic cardiovascular disease with history of myocardial infarction (DX 18, 29).

On March 8, 2004, Dr. Levinson reviewed the records, including his previous two examinations and the miner's hospital records. Dr. Levinson stated on physical examination he found the miner was not suffering from coal worker's pneumoconiosis but did have chronic obstructive pulmonary disease secondary to cigarette smoking and a right lower lobe density with right pleural effusion with suggestion of nodular masses strongly suspicious of malignancy. In addition, Dr. Levinson noted the miner had advanced arteriosclerotic cardiovascular disease with history of previous myocardial infarction and numerous hospitalizations for angina, heart failure, cardiac complications, abdominal aortic aneurysm surgery as well as peripheral vascular surgery which required a bypass. Dr. Levinson stated there was no sign of cor pulmonale or right sided heart problems related to any degree of underlying pulmonary disease.

Dr. Levinson also reviewed the miner's final hospitalization and autopsy findings as well as the autopsy review report by Dr. Oesterling. He noted the miner's history of 45 years of coal mine employment with normal pulmonary function study results at the end of his coal mine employment. In addition, he noted the miner' history of advanced arteriosclerotic cardiovascular disease which affected his heart, caused coronary artery obstruction and required multiple surgeries. In addition, Dr. Levinson noted the autopsy evidence of a very mild form of macular coal worker's pneumoconiosis. He stated it was clear this coal worker's pneumoconiosis was insufficient to have altered the miner's pulmonary function and he reiterated his earlier opinion the miner had no pulmonary symptomatology during his lifetime. Dr. Levinson stated the coal worker's pneumoconiosis present did not contribute to the miner's death. He noted there was no indication of treatment for the coal worker's pneumoconiosis in the hospital records. He stated the miner had no disability due to occupational disease. He concluded the causes of the miner's death ware clearly related to the advanced arteriosclerotic cardiovascular disease and were unrelated to the miner's coal mine employment (EX 1).

At a deposition taken on April 7, 2004, Dr. Levinson reiterated many of his written findings. In discussing the pulmonary function study results, Dr. Levinson stated the fact the miner's pulmonary function study results fluctuated showed the lower values were not due to coal mine employment but rather he felt the miner demonstrated better effort on tests with better results. Dr. Levinson stated there was no worsening of the miner's pulmonary function from 1997 to 2000. Dr. Levinson noted again that the miner had chronic obstructive pulmonary disease and emphysema due to cigarette smoking based on physical examination findings, chest x-ray, and pulmonary function study results. Dr. Levinson noted his examination in 1997 and 2000 confirmed the significant weight loss noted in other medical records. He stated the miner's weight loss was due to systemic deterioration in cardiac cachexia or cardiac failure or a possible malignancy. On review of the autopsy, he stated the measurements did not establish cor pulmonale and he stated Dr. Bindie, the autopsy prosector, did not diagnose cor pulmonale. He stated the coal mine dust present on autopsy was not sufficient to have compromised the miner's pulmonary status. Dr. Levinson again noted the normal pulmonary function study results in 1991 at the end of 45 years of coal mine employment. Based on the degree of coal worker's pneumoconiosis present, it did not cause, contribute to or hasten the miner's death. Rather, the

miner's death was due to advanced arteriosclerotic cardiovascular disease with multiple coronary artery obstructions, development of abdominal aortic aneurysm, and an aneurysm at the previous graft location which required surgery. The miner's post-operative condition included shock and hypotension (EX 5).

On March 22, 2004, Dr. R. Kraynak stated he had been the miner's treating physician since March 10, 1993. He stated the miner had shortness of breath due to Black Lung Disease and had a progressive downhill course. Dr. Kraynak reviewed the hospital records, the death certificate, and the autopsy findings. He stated the miner was totally disabled by coal worker's pneumoconiosis and he stated coal worker's pneumoconiosis was a substantial contributing factor in the miner's death. Dr. Kraynak stated the miner would have been in a much better position to fight off these diseases absent coal worker's pneumoconiosis and would have been in a better position to oxygenate his blood. He stated the miner's respiratory system was a burden on the already diseased heart (CX 1).

At a deposition taken on March 25, 2004, Dr. Kraynak stated he did not consult with the physicians treating the miner's extensive cardiac problems. He last saw the miner in June, 2000 and the miner was worse on this visit. Thus, he stated the miner was in a poor position to survive the surgery on the aneurysm and he developed arrhythmias. Dr. Kraynak stated the pulmonary hypertension which was caused by chronic obstructive pulmonary disease and interstitial lung disease and it contributed to the miner's heart failure. Dr. Kraynak stated the miner's emphysema was due to smoking and coal mine employment and the coal worker's pneumoconiosis was due to coal mine employment. He also stated he disagreed with Dr. Oesterling's opinion that the miner's pulmonary hypertension was due to cardiac disease. Dr. Kraynak stated the miner's history of smoking 45 years was not that different that the 30 to 35 years he considered in making his opinion. This change did not affect his opinion regarding the etiology of the miner's lung condition or the cause of the miner's death (CX 3). The notes from Dr. Kraynak's office visits with the miner are very brief. Eight pages include the notes for the thirty-eight visits over this nine year period (CX 50).

The miner was hospitalized several times including May, 1993, February, 1995, January, 1998, April, 1999, May, 1999, January, 2000, and the final hospitalization from August 30 to his death on September 2, 2000. Hospital records indicate the miner was treated for various cardiac problems, including premature ventricular beats, coronary artery disease, myocardial infarction, peripheral vascular disease, hypertension, femoral artery occlusion with bypass graft, congestive heart failure, and coronary artery bypass surgery. These records often list anthracosilicosis with pulmonary emphysema or coal worker's pneumoconiosis in the list of diagnoses. The records do not indicate any treatment, however, for the coal worker's pneumoconiosis. The discharge diagnosis for the final hospitalization includes: 1) ruptured femoral aneurysm at anastomotic site, left leg; 2) occlusion of left superficial femoral artery; 3) compensated congestive heart failure; 4) chronic obstructive pulmonary disease; 5) atrial fibrillation; 6) ventricular tachycardia; 7) coal worker's pneumoconiosis; 8) hypertension; 9) coronary artery disease; and 10) status post coronary artery bypass graft. The records indicate various tests indicated a variety of problems including prominent cardiomegaly, left ventricular enlargement, atrial fibrillation, complete occlusion of right superficial femoral artery, large pseudoaneurysm of left graft anastomosis, and moderate size right pleural effusion. Surgery was performed on the ruptured femoral aneurysm

on September 1, 2000. Following surgery, the miner received blood transfusions and remained in ICU. On September 2, 2000, he developed ventricular tachycardia and resuscitative efforts were not successful. The miner died on September 2, 2000 (DX 45, 47, 48, 49).

The death certificate, signed by Charles Begansky, Deputy Coroner, stated the miner's death was due to sudden carcinogenic shock secondary to cardiac dysrythmia and ventricular tachycardia due to coronary artery disease. The death certificate listed other significant conditions which included pulmonary emphysema and simple coal worker's pneumoconiosis (DX 23).

Dr. R. Bindie conducted an autopsy of lungs on September 2, 2000. Dr. Bindie reported on gross examination of the miner's lungs bullous emphysematous changes were evident in both upper lobes and there was moderate to severe confluent subpleural anthracosis. There was anthracosis of the pulmonary hilar lymph nodes and on sectioning the lungs, scattered throughout all lobes of both lungs were soft non-palpable black macular structures averaging 0.5 to 0.6 in diameter. There were also diffuse emphysematous changes. On microscopic examination, Dr. Bindie reported extensive pleural fibrosis of the right lung. Scattered throughout the sections was pigmented intra-alveolar macrophages and focal calcification. In addition, scattered small peripheral fibrinous thromboemboli were present. Dr. Bindie reported collections of black anthracotic pigment about vascular structures and bronchi and in the subpleural areas focally resembling early simple coal worker's pneumoconiosis. He also reported sclerosis of pulmonary arterioles, granulomatous pneumonitis with laminated calcified structures resembling so-called blue bodies. In addition, there was chronic interstitial pneumonitis with fibrosis and emphysematous changes. Dr. Bindie also reported pulmonary hemorrhage and focal areas resembling honeycombing as well as chronic bronchitis. Dr. Bindie stated after reviewing the circumstances of death, the past medical history and medical studies, and the limited autopsy findings, the cause of death is considered as listed on the official death certificate as cardiogenic shock secondary to ventricular tachycardia and coronary artery disease with multifactorial chronic obstructive pulmonary disease considered as a substantial contributory cause of death. He noted there was emphysema, chronic interstitial pneumonitis, and simple coal worker's pneumoconiosis and right ventricular hypertrophy consistent with a diagnosis of cor pulmonale. Dr. Bindie's final list of autopsy findings included pulmonary emphysema, extensive, bullous and diffuse, chronic interstitial pneumonitis with fibrosis and honeycombing, anthracosis and early simple coal worker's pneumoconiosis, granulomatous pneumonitis associated with pulmonary blue bodies, calcification of the lung, focal, peripheral pulmonary thromboembolia, fibrinous, sclerosis, pulmonary arterioles, pulmonary congestion and edema, lungs bilateral and moderate, hemosiderosis and siderosis in the lungs, pleural adhesions, bilateral, fibrosis encapsulation, right lung, right ventricular hypertrophy, myocardial fibrosis, focal, pleural effusion, serosanguinous, right, slight, and anthracosis, thoracic lymph nodes (DX 34).

Dr. E. Oesterling, Sr., a board certified pathologist, reviewed the evidence and reviewed the histologic slides on July 22, 2003. His written report included a lengthy and thorough discussion of his findings on the slides. In addition, Dr. Oesterling included photomicrographs taken of the slides to illustrate his discussion. Based on his examination of the slides, Dr. Oesterling reached the following conclusions: 1) minimal evidence of coal worker's pneumoconiosis in the form of mild macular disease process; 2) extensive evidence of

intrapulmonary hemorrhage with resultant diffuse early hemosiderosis of lung tissue; 3) extensive vascular change within the lung sections in the form of pulmonary plexogenic arteriopathy; 4) evidence of capillary hemangiomatosis, proliferation of capillaries within the intersitium (this process is unrelated to coal mine dust exposure, but is related to pulmonary hypertension); 5) evidence of pulmonary hypertension demonstrated by muscular hypertrophy within the pulmonary arterial tree; and 6) evidence of prominent blue body deposition throughout the lung sections (a nonspecific change commonly seen in interstitial pulmonary change). Dr. Oesterling cited a medical text which discussed the causes of intrapulmonary hemorrhage which are consistent with the findings he noted on examination of the lung slides. In conclusion, Dr. Oesterling stated the following: 1) a very mild form of macular coal worker's pneumoconiosis is present; 2) the level of disease present was insufficient to have altered pulmonary function, thus, it would not have produced lifetime symptomatology nor would it have in any way contributed to, hastened or caused the miner's death; 3) lifetime pulmonary problems were secondary to pulmonary emphysema which resulted from a significant cigarette smoking history; 4) the miner had a significant lifetime history of severe arteriosclerotic coronary vascular disease with cerebral, cardiac and peripheral complications; and 5) the cardiac disease produced marked passive pulmonary congestion with further complications resulting in diffuse intra-alveolar hemorrhage, prominent hemosiderosis and extensive pulmonary fibrosis which were not related to coal mine dust exposure (DX 51).

At a deposition taken on April 8, 2004, Dr. Oesterling discussed his pathological findings in great detail. He noted of the sixteen slides, twelve were from the lungs, two were from the right ventricular heart muscle, one was from the bronchus and one was from a lymph node. Dr. Oesterling reiterated his written finding that the slides were well sectioned and well sustained. Dr. Oesterling explained in great detail that the findings of macular changes with minimal finding of black pigment with no host response. The macules present represent little of no functional implication. Since the silica is fibrinogenic, it will produce fibrosis so on examination one looks for a fibrotic core, collagen or a central nodule if the disease has progressed to the nodular stage, either micro-nodular or macro-nodular. Here there were no nodules nor any evidence of the host response except for the dust deposited as macules. Dr. Oesterling noted again other changes as listed in his written report, including the hemosiderin and blood in the lymph node. These demonstrated the miner had bleeding in his lung tissue which was not from coal mine dust exposure but was from intrapulmonary hemorrhage. Dr. Oesterling stated the intrapulmonary hemorrhage was due to vasculitis, pulmonary hypertension and the initiation of capillary changes. The miner had significant vascular disease and multiple surgeries on his arteries. The significant cardiovascular disease caused passive congestion in his lungs which caused more work for the right side of his heart. The miner had pulmonary hypertension and biventricular enlargement of his heart. These conditions resulted in intravascular clotting which further impeded his blood flow through the lung which caused more pulmonary hypertension and more strain on the right side of the heart. The changes in the right side of the miner's heart, therefore, were not due to coal dust exposure but to the cardiovascular disease. Finally, the honeycombing present was due to the emphysema, alveolar damage due to hemorrhage in the lung, and scarring from the hemosiderin which destroyed the normal lining of the cells. None of these causes are due to the miner's coal mine employment or coal dust exposure. Dr. Oesterling reiterated the miner had intrapulmonary hemorrhage which was due to passive pulmonary congestion and hemosiderosis which caused pulmonary hypertension, capillary hemangiomatosis

and hemo-occlusive disease. None of these conditions was related to his coal dust exposure. The cause of his pulmonary hypertension was his cardiovascular disease. Dr. Oesterling noted in the hospital the miner was not treated for coal worker's pneumoconiosis. The significant emphysema based on the autopsy report was a cause early on of the miner's pulmonary symptoms, however, the heart disease and secondary changes became more significant in the months before he died. The miner's death was due to cardiovascular disease and complications. The fibrosis present did not contain silica, so it was due to other causes and not to coal dust exposure (EX 3).

On February 2, 2004, Dr. M. Wick, reviewed the medical records and the lung slides. Dr. Wick reported the slides showed evidence of marked centrilobular emphysema, chronic pulmonary thromboembolic disease with secondary arterial plexogenesis; alveolar and interstitial hemosiderosis, diffuse pulmonary anthracosis with the focal presence of interstitial polarizable material consistent with silica. The focal linear interstitial pulmonary fibrosis was present without nodule formation. There were no pleural plaques, pleural calcification or asbestos bodies present. In addition, there was no malignancy in the lungs. The sections of the myocardium demonstrated multifocal interstitial sclerosis consistent with ischemic cardiomyopathy. Dr. Wick stated these findings indicate the principal clinically relevant lung disease was causally related to cigarette smoking, heart failure, and thromboembolic events. The mild (macular) form of pulmonary anthracosilicosis present would have produced no functional abnormality during life and is only a morphological diagnosis (EX 2).

Dr. D. Prince, a pulmonary specialist, reviewed the records on March 22, 2004. Dr. Prince stated that based on the moderately severe airflow obstruction with prominent arterial oxygen desaturation on physical activity the Claimant was totally disabled during his lifetime from coal mine employment. The autopsy showed significant changes of pulmonary arterial hypertension. Dr. Prince stated the miner's death actually occurred due to underlying pulmonary hypertension precipitating ventricular arrhythmias and right ventricular failure. Dr. Prince noted there is clear pathological evidence of coal worker's pneumoconiosis. He stated the pulmonary hypertension occurred secondary to hypoxemia from chronic lung disease. The chronic exercise-induced hypoxemia was due to pulmonary impairment from chronic obstructive pulmonary disease due to smoking and coal worker's pneumoconiosis due to coal mine employment. Dr. Prince stated studies have shown complicated pneumoconiosis is not necessary for significant airflow obstruction. Thus, coal dust was a contributing factor to the underlying obstructive airways disease, exercise-induced hypoxemia, and pulmonary hypertension and, therefore, was clearly a significant contributing factor in the miner's death (CX 5).

DISCUSSION AND CONCLUSIONS — MINER'S CLAIM

The miner's claim was filed December 3, 1999 (DX 1). Therefore, entitlement to benefits must be established under the regulatory criteria at Part 718. To be entitled to benefits under Part 718, a claimant must establish by a preponderance of the evidence that (1) he suffers from pneumoconiosis; (2) the pneumoconiosis arose out of coal mine employment; (3) he is totally disabled; and (4) his total disability is caused by pneumoconiosis. *See Gee v. W.G. Moore & Sons,* 9 BLR 1-4 (1986). Failure to establish any of these elements precludes recovery under the Act.

In addition, since the miner's prior claims were denied, this most recent claim must also be denied on the grounds of the earlier denial, unless the claimant demonstrates a material change in conditions since the date upon which the order denying the prior claim became final. 20 CFR § 725.309(d)[3]. As noted above, Judge Brown found the evidence was sufficient in the prior claim to establish the presence of pneumoconiosis which arose from coal mine employment. Pursuant to Section 725.309(d), since Claimant failed to establish total disability due to pneumoconiosis in the prior denial, however, benefits will be awarded only if Claimant has demonstrated a "material change in condition". In assessing whether the miner has demonstrated a "material change in conditions", the Third Circuit has held the administrative law judge should consider all of the new evidence, favorable and unfavorable, and determine whether the miner has proven at least one of the elements previously adjudicated against him. *LaBelle Processing Co. v. Swarrow,* 72 F.3d 308 (3rd Cir. 1995). Since Claimant failed to establish total disability due to pneumoconiosis in his two previous claims, the newly submitted evidence will be reviewed to determine if Claimant has now established that one of those elements.

Total Disability

The determination of the existence of a totally disabling respiratory or pulmonary impairment shall be made under the provisions of Section 718.204. A claimant shall be considered totally disabled if the irrebuttable presumption of Section 718.304 applies to his claim. 20 CFR § 718.204(b)(1). Where, as here, if the irrebuttable presumption does not apply, a miner shall be considered totally disabled if he is prevented from performing his usual coal mine work or comparable and gainful work. In the absence of contrary probative evidence, evidence which meets one of the Section 718.204(b)(2) standards shall establish the claimant's total disability. According to Section 718.204(b)(2), the criteria to be applied in determining total disability include: 1) pulmonary function studies, 2) arterial blood gas tests, 3) a cor pulmonale diagnosis and 4) a reasoned medical opinion concluding total disability.

Pulmonary Function Studies

Pulmonary function study results were submitted for evaluation on the issue of total disability under Section 718.204(b)(2)(i). Assessment of these results is dependent on Claimant's height which was recorded between sixty-seven and sixty inches. The fact-finder must resolve conflicting heights of the miner on the ventilatory study reports in the claim. *Protopappas v. Director, OWCP*, 6 B.L.R. 1-221 (1983). This is particularly true when the discrepancies may affect whether or not the tests are "qualifying." *Toler v. Eastern Associated Coal Co.*, 42 F.3d 3 (4th cir. 1995). I find the miner is sixty-three and one-half inches here, his average reported height. None of the newly submitted pulmonary function study reports demonstrated qualifying values. I find Claimant has not established total disability under the provisions of subsection 718.204(b)(2)(i).

Arterial Blood Gas Studies

[3] This claim was pending prior to January 19, 2001, the date new regulations became applicable. 20 CFR 725.2 provides that the new regulation at Section 725.309 does not apply to adjudication of claims pending on January 19, 2001. Therefore, the prior regulation at Section 725.309 will be applicable to this claim.

Three new arterial blood gas studies were submitted subsequent to the miner's third claim for benefits. On the test taken on January 21, 2000, the miner demonstrated qualifying values on the January 21, 2000 blood gas study, but non-qualifying values on the March, 1997 and May, 2000 blood gas study. The January, 2000 study would be sufficient to establish total disability under the provisions of subsection 718.204(b)(2)(ii) if credited.

Cor Pulmonale

A claimant may also establish total disability by providing medical evidence of cor pulmonale with right-sided congestive heart failure pursuant to Section 718.204(b)(2)(iii). Although Dr. Bindie mentioned cor pulmonale in his autopsy findings, I am more persuaded by Dr. Oesterling's discussion of the cardiac changes in this case. Dr. Oesterling explained in detail how the miner's serious and advanced cardiac disease caused left ventricular failure which led to passive congestion in the miner's lungs and, thus, caused a greater strain on the right side of the miner's heart. Since the miner had biventricular enlargement (or enlargement of both the left and right side of his heart), Dr. Oesterling explained the changes on the right side of his heart were related to his advanced cardiovascular disease and not to pulmonary problems. Therefore, I find the evidence fails to establish cor pulmonale and total disability due to pneumoconiosis.

Medical Opinions

The remaining means of establishing a totally disabling respiratory or pulmonary impairment under Section 718.204(b)(2) is with a reasoned medical opinion which concludes total disability is present, if the opinion is based on medically acceptable clinical and laboratory diagnostic techniques. A claimant must demonstrate that his respiratory or pulmonary condition prevents him from engaging in his "usual" coal mine employment or comparable and gainful employment". 20 C.F.R. §718.204(b)(2)(iv).

On consideration of the newly submitted medical opinion reports, I accord greatest weight to those reports which considered the autopsy findings as well as the medical treatment and hospital records. Thus, I accord less weight to the medical opinions of Drs. Mariglio and Fino whose opinions on the miner's pulmonary capacity were written without the more complete medical information provided by the autopsy and final hospitalization reports.

I also accord little weight to Dr. Kraynak's opinions. Dr. Kraynak's examination findings were very brief, he summarized almost forty visits on eight pages of notes, while Dr. Levinson wrote three pages of notes related to one examination. Furthermore, I find Dr. Kraynak's opinions regarding the miner's pulmonary condition outweighed by Dr. Oesterling's more persuasive consideration of the autopsy evidence, as discussed in detail below. Dr. Bindie did not discuss the miner's pulmonary capacity prior to his death in the autopsy report.

Dr. Levinson concluded the miner was not disabled by the coal worker's pneumoconiosis present on autopsy since he stated, based on Dr. Oesterling's review, it was insufficient to have altered the miner's pulmonary function, and there was no symptomatology during his lifetime. Dr. Wick also agreed the miner's mild macular form of pulmonary anthracosilicosis caused no

functional abnormality during his life, however, Dr. Wick did not discuss the basis for this conclusion. Dr. Prince concluded the changes on pulmonary testing in the miner's last year of his life were due to pneumoconiosis and established total disability due to pneumoconiosis.

Dr. Oesterling, as noted above, provided the most thorough and detailed discussion of the findings on autopsy. He included specific findings on autopsy to support his conclusion that the pneumoconiosis present was too minimal to have altered the miner's pulmonary function. Specifically, Dr. Oesterling noted the pneumoconiosis present was only in macular form, had not caused any fibrosis or nodules. Dr. Oesterling's specific and thorough discussion of the anatomical evidence is also credible in light of his high qualifications as a board certified pathologist. I note his opinion is well supported by the fact that the miner's pulmonary laboratory test results were non-qualifying until March, 2000, only six months prior to his death from cardiovascular disease. Under these circumstances, I find Dr. Oesterling's findings reliable, well supported and well reasoned. I find his opinion outweighs the contrary opinions of record which are not as well supported, as well reasoned nor as credible. Accordingly, I find the medical opinion evidence does not establish total disability due to pneumoconiosis under Section 718.204(b)(2)(iv) and (c).

In summary, the evidence does not establish the miner was totally disabled due to pneumoconiosis by pulmonary function study, blood gas study, by the presence of cor pulmonale or by medical opinion reports. I find, therefore, total disability due to pneumoconiosis has not been established under the provisions of Section 718.204. Since the evidence does not establish this element of entitlement which was adjudicated against the miner in the prior denial, I find this claim shall be denied on the basis of the prior denial as set forth in Section 725.309(d). Accordingly, the miner's claim for benefits filed on December 3, 1999 shall be denied.

DISCUSSION AND CONCLUSIONS — WIDOW'S CLAIM

Claimant-widow filed her claim on January 3, 2002 (DX 41). Therefore, entitlement to benefits must be established under the regulatory criteria at Part 718. Section 718.205(a) provides that benefits are available to eligible survivors of a miner whose death was due to pneumoconiosis which arose out of coal mine employment. An eligible survivor will be entitled to benefits if claimant proves that: 1) The miner had pneumoconiosis; 2) The miner's pneumoconiosis arose out of coal mine employment; and 3) The miner's death was due to pneumoconiosis as provided by Section 718.205. For purposes of claims filed after January 1, 1982, death will be considered due to pneumoconiosis if any of the following criteria is met: 1) Where competent medical evidence establishes that pneumoconiosis was the cause of the miner's death; or 2) Where pneumoconiosis was a substantially contributing cause or factor leading to the miner's death; or 3) Where the presumption set forth in § 718.304 (evidence of complicated pneumoconiosis) is applicable. 20 C.F.R. 718.205(c). The regulations also provide that survivors are not eligible for benefits where the miner's death was caused by a traumatic injury or the principal cause of death was a medical condition not related to pneumoconiosis, unless the evidence establishes that pneumoconiosis was a substantially contributing cause of death. 20 C.F.R. 718.205(c)(4). The regulations further provide that pneumoconiosis is a "substantially contributing cause" of a miner's death if it hastens the miner's death. 20 C.F.R. 718.205(c)(5).

As noted above, the presence of pneumoconiosis was established in the miner's claim and has been confirmed by the autopsy findings as well as the probative and persuasive pathological review report of Dr. Oesterling. Accordingly, I find the presence of pneumoconiosis which arose out of coal mine employment has been established. Therefore, the Claimant-widow's entitlement to benefits on her survivor's claim is dependent upon her meeting her burden of establishing that the miner's death was due to coal workers' pneumoconiosis. 20 C.F.R. §718.205.

The medical reports are set forth above. On consideration of the medical evidence, I accord greatest weight to Dr. Oesterling's medical opinion. While I note Dr. Bindie stated chronic obstructive pulmonary disease was a substantially contributing cause of death, he did not discuss if he considered the etiology of the chronic obstructive pulmonary disease to include coal mine dust exposure or only the miner's extensive and long cigarette smoking history. In addition, while he clearly found simple coal worker's pneumoconiosis present, he did not state that the coal worker's pneumoconiosis present contributed to the miner's death. As noted above, I found Dr. Oesterling's thorough and detailed discussion of findings on the lung slides as related to the cause of the miner's death probative and persuasive. Dr. Oesterling's discussion of the miner's advanced cardiovascular disease and the resultant impact on the miner's lungs was well reasoned and well supported as demonstrated by the pathological findings he described. Dr. Oesterling's explanation of the cause of pulmonary hypertension was supported by the medical text and clearly outweighs Dr. Kraynak's unsupported contradictory conclusion. Dr. Oesterling's opinion and detailed explanations also outweigh Dr. Prince's findings which are not as thorough nor as well supported by either findings on autopsy, the lung slides or by the medical texts. In addition, Dr. Oesterling's discussion of the specific findings on autopsy of macular simple coal worker's pneumoconiosis but no fibrosis or nodularity provides a clear and well supported basis for his conclusion the pneumoconiosis present was insufficient to have altered the miner's pulmonary function or to have contributed to his death.

No physician concludes the coal worker's pneumoconiosis present caused the miner's death. All the physicians agreed the miner's death was due to his serious and advanced cardiovascular disease. Thus, death due to pneumoconiosis is not established under subsection 718.205(c)(1).

For the reasons set forth above, I find Dr. Oesterling's opinion also establishes that pneumoconiosis was not a factor in nor did it contribute to or hasten the miner's death. Thus, death due to pneumoconiosis is not established under subsection 718.205(c)(2). Furthermore, the presumption at Section 718.205(c)(3) is not applicable since the autopsy clearly did not establish the presence of complicated pneumoconiosis. Accordingly, I find Claimant has not established the miner's death was due to pneumoconiosis as required by Section 718.205(c).

As Claimant has not established that the miner's death was due to pneumoconiosis under the provisions of Section 718.205(c), she is not entitled to survivor's benefits under the Act.

ORDER

The claim for benefits by Frederick J. Israel, deceased, shall be **DENIED**.

The claim of Marion Israel, widow of Frederick J. Israel, deceased miner, for survivor's benefits is hereby **DENIED**.

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PAUL H. TEITLER
Administrative Law Judge

Cherry Hill, New Jersey

NOTICE OF APPEAL RIGHTS: Pursuant to 20 C.F.R. 725.481, any party dissatisfied with this Decision and Order may appeal it to the Benefits Review Board within 30 (thirty) days from the date of this Decision by filing a Notice of Appeal with the Benefits Review Board at P.O. Box 37601, Washington, D.C. 20018-7601. A copy of this notice must also be served on Donald S. Shire, Associate Solicitor, Room N-2605, 200 Constitution Avenue, N.W., Washington, D.C. 20210.